

If a Population Crashes in Prehistory, and There Is No Paleodemographer There to Hear It, Does It Make a Sound?

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ABSTRACT Catastrophic episodes (e.g., epidemics, natural disasters) strike with only limited regard for age. A large percentage of catastrophic mortality in a population can lead to a death distribution that resembles the living distribution, which includes greater numbers of older children, adolescents, and young adults than typical mortality profiles. This paper examines both the population implications of a large catastrophic mortality event, based on the Black Death as it ravaged medieval Europe, and its long-term effects on age-at-death distributions. An increased prevalence of epidemic disease is a common feature of reconstructions of the shift to agriculture and the rise of urban centers. The model begins with a hypothetical Medieval living population. This population is stable and characterized by slow growth. It has fertility and mortality rates consistent with a natural-fertility, agrarian population. The effects of catastrophic episodes are simulated by projecting the model population and subjecting it to one large (30% mortality) catastrophic episode as part of a 100-year population projection. A pair of Leslie matrices forms the basis of the projection. The catastrophic episode has important, long-term effects on both the living population and the cumulative distribution of death. The living population fails to recover from plague losses; at the end of the projection, population is still less than 75% its pre-plague level. The age-at-death distribution takes on the juvenile-young adult-heavy profile characteristic of many archaeological samples. The cumulative death profile based on the projection differs from that produced by the stable model significantly ($P < 0.05$) for 25–50 years after the plague episode, depending on sample size. *Am J Phys Anthropol* 112:181–190, 2000.

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Contemporary human populations display regular patterns of mortality (Brass, 1971; Coale and Demeny, 1966, 1983). The risk of death is high near birth and then falls dramatically, reaching its lowest point in early adolescence. It then climbs steadily through adulthood as we senesce. Excavated skeletal series, such as the early Medieval series from Bonaduz, Switzerland (Brunner, 1972; see Fig. 1), frequently exhibit different patterns characterized by greater percentages of death among older

children, adolescents, and young adults (Acasadi and Nemeskeri, 1970; Lovejoy et al., 1977; Weiss, 1973). Paleodemographers (e.g., Howell, 1982; Keckler, 1997; Lovejoy et al., 1977; Meindl and Russell, 1998; Milner et al., 1989; Paine, 1989, 1997; Wood et al., 1992) have long debated the meaning

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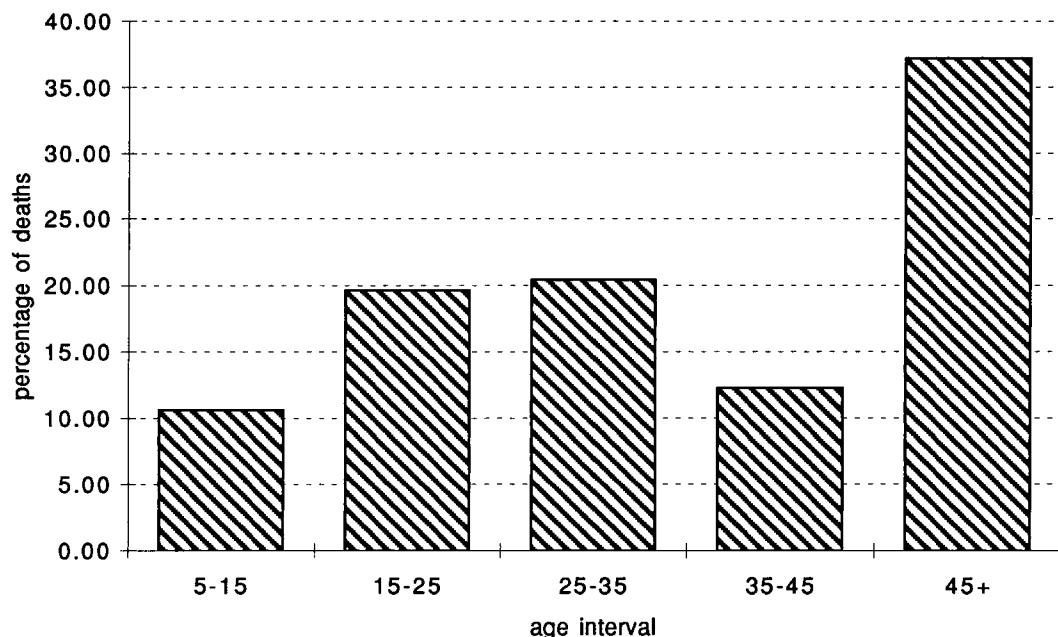


Fig. 1. Distribution of age-at-death for the Swiss Medieval site of Bonaduz (Brunner, 1972), grouped in 10-year age intervals. Individuals under 5 years old have been omitted.

and significance of these patterns. Some (e.g., Lovejoy et al., 1977; Meindl and Russell, 1998; Storey, 1992) have argued that these patterns imply fundamental differences between prehistoric and recent populations and have questioned whether using contemporary populations to understand the prehistoric record is justifiable. Others (e.g., Howell, 1982) have used patterns of human biology from contemporary populations (e.g., age-specific fertility and mortality rates) to contest paleodemographic interpretations. This paper follows an argument I have made elsewhere (e.g., Paine, 1997): to make robust conclusions from paleodemographic data, we must develop explicit models of how cultural and biological processes may manifest themselves in the skeletal record. The goal of such models is to *explain* variation in age-patterns-of-death, not to dismiss it. This paper provides a specific case example, the long-term effects of a catastrophic mortality event, to show one way such a model could be developed and tested.

The present paper has three main goals. The first is to demonstrate one means of modeling the long-term effects of demographic events. I have chosen a Leslie ma-

trix projection for this exercise, but many other techniques are available (for an excellent alternative, see Keckler, 1997). My second goal is to model the general effects of a large catastrophic event on both a living population and its age-at-death distribution. The third goal is more specific to paleodemography: to examine how long such an event might be visible in the age-at-death distribution of a cemetery before it is subsumed by a normal pattern of attritional mortality.

For the purposes of this exercise, a catastrophic episode, whether caused by natural or human-based calamity, is defined as a short-term mortality crisis that strikes populations with limited or no regard for age or sex. A large percentage of catastrophic mortality in a population leads to a death distribution that resembles the living distribution (Keckler, 1997). This paper examines the effects of large catastrophic events using a simplified model of an important catastrophic event in world history, the Black Death, on a hypothetical Medieval living population and on its distribution of death. The effects of the plague are modeled as a single, large episode of catastrophic mortal-

TABLE 1. *Leslie matrix for a mythical animal, uniformitarianus fictiva*

Leslie =	0.0	0.4	0.6	0.4
	0.8	0.0	0.0	0.0
	0.0	0.9	0.0	0.0
	0.0	0.0	0.7	0.0

ity that is part of a 100-year population projection (theoretically from AD 1347–1446). Though the model was created based on characteristics of the Black Death, it is intentionally generalized, and could be used as a starting point for a variety of more sophisticated models. Future exercises will test more elaborate models, examining the effects of multiple episodes, varying severity of events, and heterogeneous resistance to the catastrophic episodes (whether through acquired immunities, or sociocultural variables) on living populations and on age-at-death distributions.

METHODS

The Leslie matrix

Leslie matrices (Leslie, 1945) are used to project populations forward in time. They project both population size and age structure and can be used to create full life tables and to estimate vital rates for projected populations. A Leslie matrix is square; the number of rows and columns in the matrix equals the number of age intervals. The top row of the matrix is comprised of age-specific fertility rates (ASFRs). Age-specific survival rates are found on the subdiagonal. When a Leslie matrix is postmultiplied by a vector representing an existing population, it yields a new vector representing the population at the end of the projection interval (for a good, brief review of matrix algebra, see Caswell, 1989; Leslie matrices are very clearly presented in Bradley and Meek, 1986, and in Caswell, 1989).

Table 1 is a Leslie matrix representing survivorship and fertility rates for a mythical animal, *Uniformitarianus fictiva*. This creature has different life history characteristics from humans. It has a lifespan of just 4 years, and females are fertile until death. As the top row of the matrix shows, females of this species do not reproduce in the first year of life (age intervals could represent any size interval, e.g., days, minutes, sec-

TABLE 2. *Demographic characteristics of the model stable population used in the projection¹*

α	β	e_0	TFR	CBR	CDR	r
0.65	0.95	22.05	6.1	45.23	44.49	0.00074

¹The first two columns are parameter values for the Brass (1971) relational model life tables. The other demographic values were derived from the model life table.

Parameter α in the relational life table models of Brass (1971) shifts the model mortality curve up or down, raising or lowering overall mortality. The range of reasonable values for α is from +0.8 to -1.5. Higher values (>0.00) indicate high mortality, compared to the standard.

The β parameter of Brass (1971) rotates the model mortality curve around a fixed point (51 years), making it more or less steep. Lower values decrease the slope, giving the model high infant and child mortality, compared to the standard. e_0 , life expectancy at birth; TFR, total fertility rate; CBR, crude birth rate; CDR, crude death rate; r, intrinsic rate of growth.

onds). Age-specific fertility peaks in year 3. The sum of the ASFRs in the top row is the gross reproductive rate (GRR), 1.4 in this example. The matrix is based on a 50:50 sex distribution. Individuals have an 80% probability of surviving the first year of life. Ninety percent of those who survive year 1 survive year 2, and so on.

Projecting any Leslie matrix that meets a set of very loose conditions¹ produces a stable age distribution with stable demographic rates, regardless of the initial population. The number of intervals required to approach stability varies with the size of the matrix (the number of intervals of lifespan). The *Uniformitarianus* matrix produces a stable age distribution after 5–10 intervals. The resulting stable population has a negative intrinsic growth rate (approximately -0.015), a crude birth rate (CBR) of approximately 320, and a crude death rate (CDR) of approximately 335.

Each matrix used in the Black Death projection is for single-year intervals up to age 90. It therefore contains 8,100 cells and is impractical to portray here. The mortality and fertility characteristics of the initial population are based on the standard model life tables of Brass (1971) (Standard, α = 0.65, β = 0.95). The stable population (represented by matrix S) is designed to conform to general estimates of preindustrial, agrar-

¹These conditions are set out in the theorem of Perron and Frobenius (summarized in Pollard, 1973). The primary condition is that two consecutive F_i (the age-specific fertility rates, represented in the top row of the Leslie matrix) be positive. This is true of virtually all models of human fertility.

TABLE 3. Deaths, by age, produced in a single year under the model stable conditions and in the plague year¹

Age interval ending	Stable year deaths	Plague year deaths	Equivalent in stable years
1	44,916	77,521	1.73
5	13,513	80,455	5.95
10	2,585	75,429	29.18
15	1,806	71,376	39.52
20	2,853	68,886	24.15
25	3,415	64,478	18.88
30	3,162	59,020	18.67
35	2,892	53,973	18.66
40	2,824	49,460	17.51
45	2,864	45,073	15.74
50	3,126	40,735	13.03
55	3,363	36,026	10.71
60	3,562	30,967	8.69
90	15,912	73,656	4.63

¹The rightmost column shows the number of years the stable model population would take to produce the number of age-specific deaths produced in the plague year.

ian populations (see Table 2). The model population has fertility (TFR = 6.15) consistent with a natural-fertility population (Bentley et al., 1993; Campbell and Wood, 1988). Life expectancy at birth conforms to levels suggested by paleodemographers (e.g., Storey, 1992), and the population is growing slowly.

I projected the model population for 100 years to generate a stable age distribution. This distribution represents the study population at the beginning of the projection. It is represented by a vector (v_1) used to initialize the Black Death projection. All projections were performed using Maple (version 5) for Macintosh. Maple is an algebra program for computers designed to perform complex operations. Analyses of the projected populations and all graphics were produced on a spreadsheet (Excel 4 for Macintosh). The age distribution of death (abridged) for the stable population in the final year before the plague episode is found in column 2 of Table 3.

The Black Death model

Bubonic plague (*Pasteurella pestis*) entered Europe in 1346. It arrived with a Mongol army that laid siege to the Crimean trade city of Caffa. It spread from Caffa, by ship, through the Mediterranean and to western and northern Europe (McNeil, 1977). Though the impact of the plague var-

ied by region, McNeil (1977) estimates that approximately one third of Europe's total population died of plague between 1346–1350. Most other estimates hover between 20–45%, with locally higher losses. A second wave of plague struck in the 1360s and 1370s, but is not represented in this projection. Presumably because those who had survived the first epidemic developed immunities, later death tolls were concentrated among those born after the initial wave of plague (McNeil, 1977, p. 149). Europe then fell into a century of population decline. Shortages of labor led to shrinking economies and the widespread abandonment of agricultural land. Russell (1958, quoted in McNeil, 1977) argues that the population did not recover to pre-plague levels until 1550.

To simulate the effects of the Black Death, I constructed a second Leslie matrix (C) characterized by catastrophic mortality. I began with the stable matrix and subtracted 30% from the survival rate for every age category. Hence, the survival rate for infants for a 1-year interval decreases from 0.850 to 0.550, and the survival for 18-year-olds decreases from 0.994 to 0.694. At the same time I reduced age-specific fertility rates by the same 30%. Realistically, the decline in fertility should probably be higher and should extend into the following year, but complex assumptions about fertility decline do not have a significant enough effect on the overall model to justify their inclusion.

The Black Death model is a 100-year population projection. Theoretically, the projection runs from AD 1347–1446. It is intended to show the long-term effects of the plague on a hypothetical living population, and to provide a minimal estimate of the plague's cumulative effect on the distribution of death. In the first year of the projection, the stable Leslie matrix (S) is postmultiplied by the stable population vector (v_1). This yields a normal year's worth of age-specific deaths and a new vector (v_2) representing the living population at the outset of year 2. This vector still has the age distribution characteristic of the stable population. In the second year of the projection the population experiences the plague episode. The population

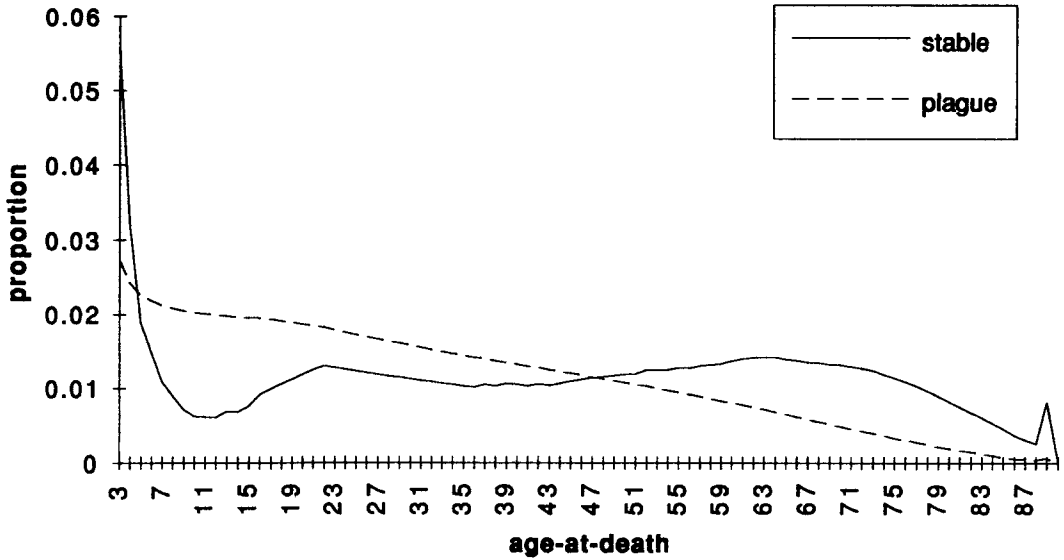


Fig. 2. Comparison of age-at-death proportions produced by the stable model population (dashed line), and by the plague model (solid line).

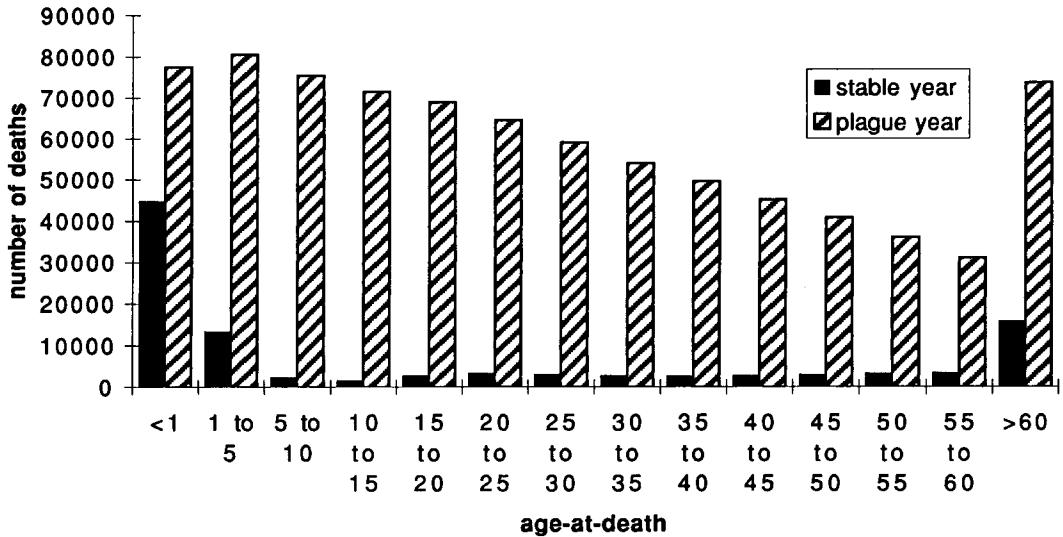


Fig. 3. Distribution of deaths produced in a single year, under the stable model conditions (solid bars), and in the plague year (grey bars).

vector (v_2) is postmultiplied by the Leslie matrix representing the plague year (C). This results in a catastrophic year's age-specific deaths and a new living age structure, represented by a new vector (v_3). The age distribution of the living population is no longer stable. During the remaining 98 years of the projection, the population is subjected to the same fertility and survivor-

ship probabilities that created the original stable population. This is accomplished by postmultiplying each new living population vector (v_i) by the stable Leslie matrix (S) and recording the new population vector and the age-specific death produced. I tracked the size and shape of the living population, yearly CBR, CDR, and r , yearly deaths, and cumulative deaths throughout

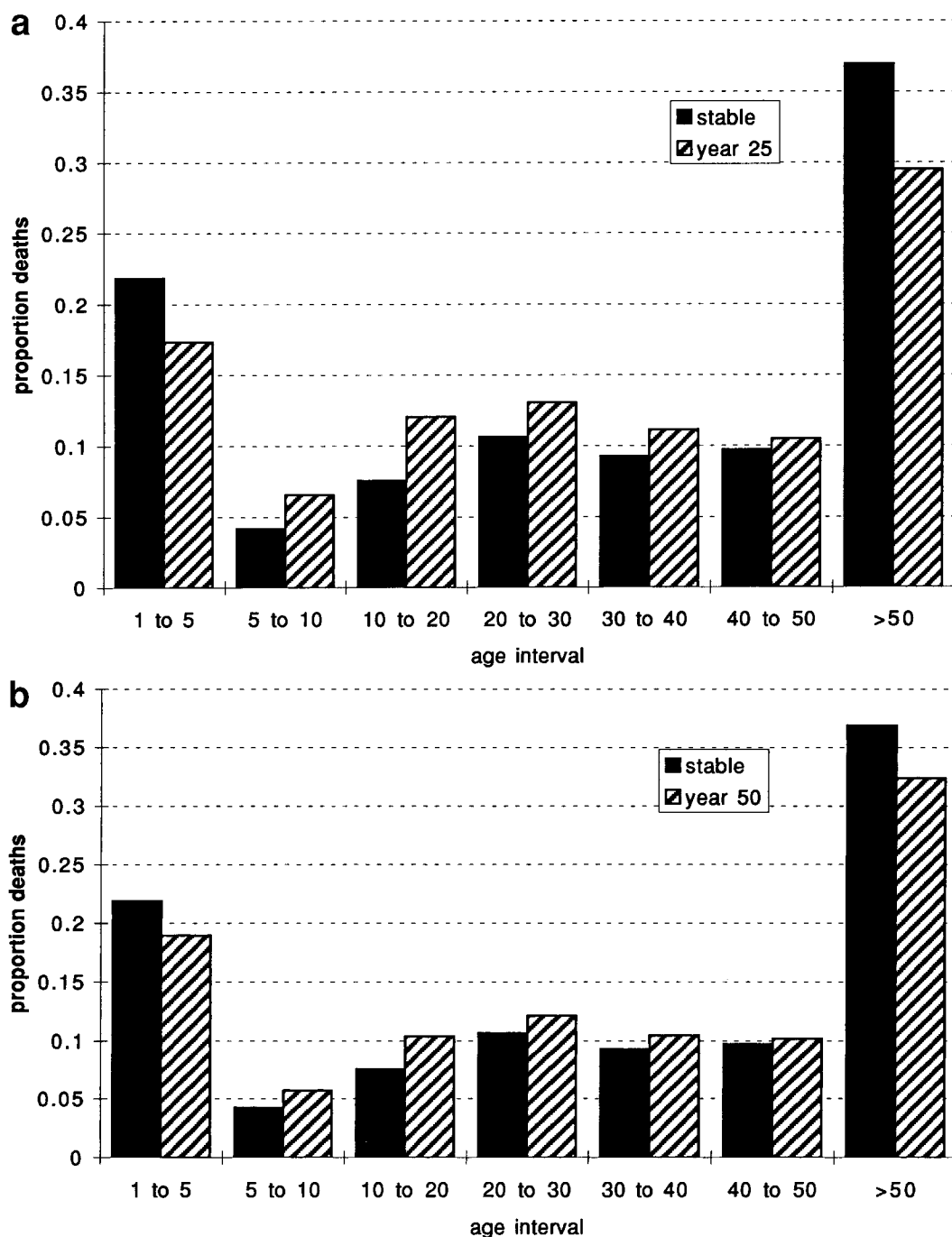


Fig. 4. Projected cumulative age-at-death distribution (grey bars) compared to the age-at-death distribution produced by the stable model population (solid bars). **a:** Cumulative age-at-death distribution 25 years after plague episode. **b:** Fifty years after plague episode. **c:** At the end of the projection, 98 years after the plague episode.

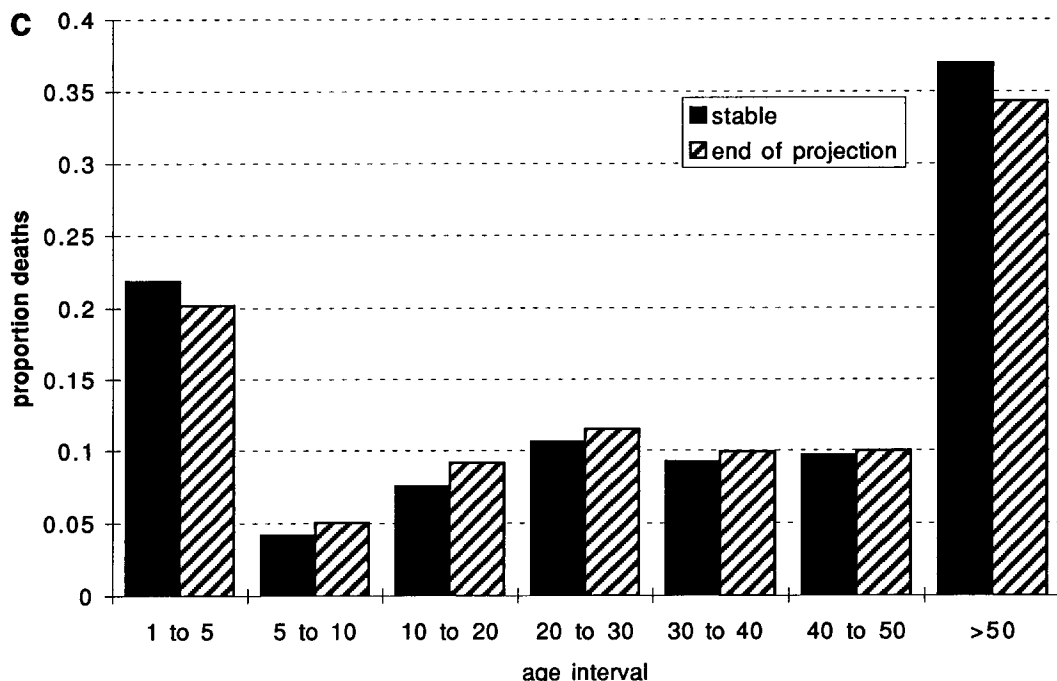


Fig. 4. (See legend page 186.)

the projection. The cumulative death distribution represents a theoretical cemetery distribution, as might be recovered by a dreaming archaeologist (with a perfectly representative sample, perfect preservation, and accurate, unbiased age and sex reconstruction).

RESULTS AND DISCUSSION

Figure 2 compares single-year death distributions left by attritional mortality under the specified stable conditions and by a year including a catastrophic mortality episode. Both were applied to the same living population (represented by v_1). Adding 30% to the risk of death, regardless of age, dramatically increases the proportion of deaths between 3–50 years of age. At the same time there is a smaller proportion of deaths after age 50. It is important to note that the simulated plague year produces more than 10 times as many overall deaths as the stable population produces in a year. This effect is magnified for ages that have low risk of death in normal years. It takes less than 2 years to produce as many infant (1 year old)

deaths as the plague year, and less than 5 years to produce as many deaths in the oldest age category (Table 3). In contrast, it takes the stable population almost 30 years to produce as many deaths between 5–10 years of age, and almost 40 years to produce as many deaths in the 10–15-year interval as in the plague year. Figure 3 compares raw deaths, by age, between a stable year and the plague year.

Over the course of the projection, the cumulative death distribution slowly converges with the stable age-at-death distribution (Fig. 4a–c). At the end of the projection, differences are still visible (Fig. 4c) but are not significant for sample sizes in the typical range of archaeological series. The length of time where statistically significant differences between the cumulative death distribution and the stable death distribution persist varies with sample size. Table 4 shows the timing of convergence for samples of 250 and 500 individuals; these would be considered good to excellent sample sizes for archaeologically-recovered skeletal series. They are equivalent to random

TABLE 4. Pearson's χ^2 test of fit between model cemetery samples (cumulative death distributions) at three points in the projection and that produced by the stable model population¹

Year	χ^2	Significance
N = 500		
25	37.208	$\alpha < 0.001$
50	14.590	$\alpha < 0.025$
100	4.776	n.s.
N = 250		
25	18.604	$\alpha < 0.005$
50	7.295	n.s.
100	2.388	n.s.

¹Sample sizes (N = 250, 500) are those of the cemetery samples, not of the living populations. n.s., no significance.

samples of size N (N = 250 or 500) from a population of unknown size. After 25 years the differences are still highly significant for samples of over 250 individuals. For samples of over 500 individuals, they are still significant after 50 years. Infants <1 year old) were not included in any comparisons because infant preservation appears to vary so widely between excavated skeletal series.

Effects of the plague on the living population

The shocks to the living population were long-lasting. A slowly growing population, like the one modeled here, requires a very long time to recover. At the end of the projection the total population was still less than 75% of its original size. The growth rate actually increases, temporarily, after the plague (Fig. 5). This happens because the numbers of high mortality, nonreproductive, young children are reduced by the plague. This temporarily boosts the crude birth rate and lowers the crude death rate. As the plague cohort of children reaches reproductive age, CBRs decline and the intrinsic growth rate drops below the stable population's r for approximately 50 years. After the Black Death, countries of northern Europe, like Denmark, experienced a long-term population crisis not unlike the one described in the projection (Benedictow, 1993; McNeil, 1977). The exact nature of these effects depends on the demographic characteristics of the original model stable population.

Though the Black Death was a particularly virulent and well-known event, it was neither the first nor the last great epidemic

to affect Europe. The Roman documentary record is filled with epidemic events, notably the Antonine plague (AD 160–165, recurrence in AD 189) and the Justinian plague (beginning AD 542). Both the Antonine and Justinian plagues resulted in similar death tolls, for the Roman world, to those from the Black Death for all of Europe. Epidemic patterns in Europe, north of the Alps, are not well-documented. Europe was less densely occupied than the Mediterranean and less well-connected to the rest of the Old World.

Effects on the distribution of death

Even at the end of the projection, the plague episode has a visible impact on the age-at-death distribution. More older-child and young-adult deaths are found than would occur in a stable population under the parameters of either Coale and Demeny (1966, 1983) or Brass (1971). At the same time there is a smaller proportion of older adult deaths than in the model stable populations. The plague events clearly bring model death distributions closer to the age-at-death distribution from Bonaduz and many other paleodemographic samples.

The projection presented here suggests that age-at-death patterns characterized by excess adolescent and young adult death, as found in the Bonaduz sample and other agrarian skeletal series, could be produced if epidemic events were part of the environment, even if they were relatively rare events. This is an interesting alternative to the argument Lovejoy et al. (1977) made to explain the mid-age bulge in the Libben skeletal series (Keckler (1997) raises similar questions). They argued that high death rates among older children and young adults reflected a low level of infectious disease, resulting in the greater survival of frail individuals through childhood and "reduced immunocompetence" at greater ages. This projection does not refute the Libben argument, but merely suggests that other possibilities should be considered. Epidemic or catastrophic events (a severe famine might have a similar effect) may have been a more important feature of New World agriculturalists' environments than previously assumed.

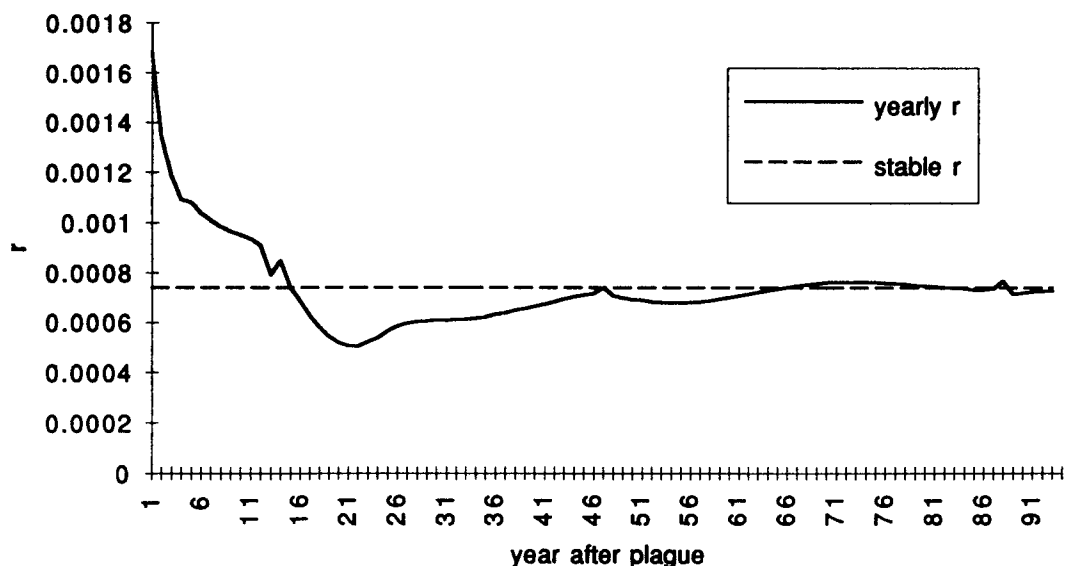


Fig. 5. Intrinsic growth rate (r), tracked by single year, following the plague episode. Dashed line represents the intrinsic growth rate of the stable model population.

This type of exercise cannot prove whether epidemic events are the cause of the adolescent/young adult bulge found in many agrarian skeletal samples. The plague projection yields patterns that are consistent with those recovered archaeologically, but does not eliminate alternative explanations. Had the projection patterns been significantly different from the archaeological pattern, this would have been an effective refutation of episodic catastrophe as an explanation of difference between the archaeological pattern and contemporary mortality patterns, like those found in the models of Coale and Demeny (1966, 1983) or Brass (1971). A similar exercise (Paine, 1997) effectively eliminated in-migration as a cause of the same pattern. If catastrophic epidemic episodes are a widespread explanation of this mortality profile (which I would consider highly premature—it may be an explanation in some cases), I would predict that this pattern should be less extreme among prehistoric foragers. Child mortality patterns from Mesolithic Europe (Paine and Boldsen, 1997) provide some tentative support of that contention, though Keckler (1997) finds evidence of catastrophic mortality patterns among prehistoric foragers. Among other factors that may lead to such a pattern, I am

particularly concerned about the effects that reference sample bias (Bocquet-Appel and Masset, 1982, 1996; Konigsberg and Frankenberg, 1992, 1994; Konigsberg et al., 1997) may have on reconstructions of age-at-death distributions.

The answer to the question posed in the title would appear to be a qualified yes. Catastrophic events like plagues do make a “sound;” they leave a clear signature on the paleodemographic record. It is a signature that paleodemographers have described in a wide range of circumstances. We need more information to distinguish whether that signature, in any given case, is the echo of an ancient catastrophe or some other as-yet unrecognized phenomenon.

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